POSTER

TCT-126

Image-Guided Beating-Heart Closure of Patent Foramen Ovale Using Novel MEMS Closure Device

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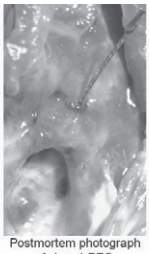
Background: Transcatheter device closure of patent foramen ovale (PFO) has received considerable attention in the past decade. The most widely used devices are deployed through the PFO channel. This approach has the disadvantage of leaving a significant amount of foreign material inside the left atrium (LA). Furthermore, the implantation of such devices may complicate future transseptal access to the LA. We have developed a novel closure system that mimics surgical PFO closure, and tested this approach.
Methods: The novel microelectromechanical systems (MEMS) device is made out of a nickel cobalt alloy using electrochemical fabrication. The device is deployed via transseptal puncture. The device has two sets of toggling wings that, after deployment, are ratcheted toward each other to the desired distance so as to bring the septum primum and secundum together. In Yorkshire pigs (n=10) a tunnel-like PFO was created under video-assisted cardioscopy and epicardial echocardiography and then closed. For PFO closure, the deployment system is advanced toward the point of septal puncture under 2D and 3D echocardiography guidance. Once the septum is penetrated, fluoroscopy is used for device deployment and ratcheting.
Results: A PFO was created in 9 animals, and 1 animal had a naturally occurring PFO. All the PFOs were successfully closed using the MEMS device. One device per animal was used in 9 cases, and, in 1 case, two devices were deployed side by side. No residual shunts were detected.

MEMS
PFO closure device



Postmortem photograph
of closed PFO,
view from the right atrium

Conclusions: Image-guided beating-heart PFO closure can be successfully achieved using a novel MEMS double toggle closure device. This approach, that mimics precise surgical PFO closure, may be a better alternative to current transcatheter PFO closure techniques because of its patient-specific adjustability and since a very limited amount of foreign material is left inside the LA.

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Overlapping Sirolimus-eluting Stent-induced Vasomotor Dysfunction of Both Conduit and Resistance Coronaries Is Accompanied by Local Inflammatory Response in a Porcine Model

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Background: Sirolimus-eluting stent (SES) has been shown to elicit coronary vasomotor dysfunction. However, the underlying mechanism remains uncertain. In this study, we investigated epicardial and intramyocardial resistance vasomotor function and inflammatory response after overlapping SES in porcine coronary arteries.
Methods: Overlapping bare metal stents (BMS) (n=12) and SES (n=12) were implanted into 12 pigs. Each pig received two pairs of identical stents, with S/A ratio of 1.1:1 and overlapping segment of 1/3 to ½ of single stent length. At 1 month, endothelial vasomotor function 5-10 mm distal to the stents, as well as TIMI frame count were performed with acetylcholine (Ach, 10^{-6}, 10^{-5} M/ml) and nitroglycerin (NTG, 400µg) infusion. The inflammatory reaction at stent region was assessed by histopathology analysis.
Results: Angiographic minimal luminal diameter, as well as percent diameter stenosis, was not significantly different between two types of stents (P>0.05). Endothelium-dependent vasomotion at distal non-stented reference segments was significantly impaired in SES, as compared to BMS. The mean coronary diameter changes with Ach 10^{-6} and 10^{-5} was -3.12± 3.63% and -5.39±5.89% for SES, and -0.34±9.77% and -0.96±12.16% for BMS (p<0.05, respectively). TIMI frame count following Ach 10^{-6} M/ml was also significantly increased in SES vs BMS (p<0.05). However, no differences in NTG-induced vasodilatation were observed between groups. The inflammatory cell infiltration, intramural fibrin deposition, as well as medial necrosis were all significantly increased for overlapped SES, as compared to BMS (p<0.05, respectively).
Conclusions: Abnormal endothelium-dependent relaxation of both coronary conduit and resistance arteries was demonstrated after overlapping SES implantation. Concomitant profound local histopathological changes may be contributors to vasomotor dysfunction.

TCT-128

Simulation of Functional Tricuspid Regurgitation using an isolated heart porcine model

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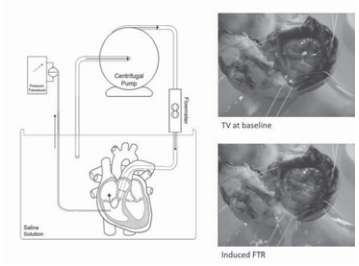
Background: Results of tricuspid annuloplasty to treat functional Tricuspid Regurgitation (FTR) are suboptimal and other therapeutic approaches should be investigated. The aim of this study is to create a reproducible "ex-vivo" bench-model of FTR in an isolated porcine heart to simulate the anatomical features of FTR during the systolic phase.
Methods: A fresh porcine heart was mounted on a rigid support into a basin full of saline. A closed-circuit including the heart was created with a centrifugal pump, equipped with connection tubes of 3/8'' of diameter (Figure). The inflow tube of the pump conveyed saline from the basin to the centrifugal pump; the outflow cannula was inserted through the pulmonary artery, across the pulmonary valve, in the right ventricle (RV) and secured with a hose clamp. Pump was activated to pressurize RV. As the RV dilated as effect of filling, TR became apparent. Regurgitant flow through the TV (TVR) was quantified by a flowmeter connected to the outflow tube. A pressure transducer was put in the RV to measure the filling pressure (RVP). A balloon was inserted in the left ventricle in order to avoid bulging of the interventricular septum towards the left when the RV was pressurized. The model was validated with 10 different porcine hearts.
Results: TVR increased proportionally with the RVP increase (TVR=0.089*RVP-1,515; R^2=0,89). Mean TVR of 0.0; 0.5; 1.0; 1.5 and 2.0 L/min were observed for RVP values of 0; 24.2±9.5; 43.2±15.6; 55.6±22.3 and 57.5±25.3 mmHg respectively.



Conclusions: The bench-model represents a reproducible system to simulate the physiopathology of the FTR and has the potential to become a standard method to evaluate new technologies to treat FTR or to perform fatigue tests of new devices. With slight modifications of the experimental setup, the concept introduced by the model can be applied to the study of other valvular diseases.

TCT-129

Better Vascular Healing Without Reduction in Anti-Restenotic Effect of a Low-dose Paclitaxel plus Simvastatin Eluting Stent: Preclinical Results

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Background: Paclitaxel-eluting-stents (PES) are consistently associated with a delayed healing of the vessel. Simvastatin-eluting-stents (IRIST®), based on a stainless-steel stent with P5® polymer, have demonstrated an endothelialization process equivalent to bare metal stents. Our objective is to analyze the response to a decrease in the amount of paclitaxel and addition of simvastatin into a combined drug-eluting-stent.
Methods: In 15 domestic swine (25 ± 3 kg), one stent per coronary artery was implanted with an intended stent-to-artery ratio of 1.1 to 1.2. Paclitaxel eluting stents (ACTIVE®, 0.36 µg/mm^2 paclitaxel) were used as controls. We tested two different formulations of simvastatin + paclitaxel-eluting-stents, both with 0.36 µg/mm^2 simvastatin + 0.13 µg/mm^2 paclitaxel (IRIST-Duo I) and 0.09 µg/mm^2 paclitaxel (IRIST-Duo II). Nine stents were assessed at 7 days to measure the quantitative rate of strut endothelialization and luminal inflammation by Scanning Electron Microscopy (SEM). The remaining 36 stents were analyzed at 28 days. Morphometric analysis was performed to calculate neointimal area (NIA), percentage of restenosis and semi-quantitative degree of endothelialization as recommended per consensus documents. Angiographic restenosis (%) and late loss (LL) were measured using off-line QCA (Medis®).
Results: Both at 7 and 28 days, significant better healing parameters were observed with IRIST-Duo II without significant differences in restenosis, neither by histomorphometry nor angiographically. The table summarizes the results:

	PES	IRIST-Duo I	IRIST-Duo II
7-days			
• Endothelialization, %	1 ±2 *	17 ±11 †	56 ±12
• Inflammation, %	23 ±8 *	13 ± 6	12 ± 2
28-days			
• Full endothelialization, %	50 *	42 †	83
• NIA, mm^2	1.4 ±0.3	2.5 ± 1.8	2.6 ±1.8
• Restenosis (histo), %	19 ±6	24 ± 14	27 ±17
• Restenosis (angio), %	10 ±8	13 ±6	15 ±6
• Late loss, mm	0.3 ±0.3	0.4 ±0.3	0.5 ±0.3
*p< 0.05 IRIST-Duo II: * vs PES; † vs IRIST-Duo I*			

Conclusions: In conclusion, lower doses of paclitaxel combined with simvastatin in a drug-eluting-stent improve the healing response without a significant compromise in anti-restenotic efficacy in preclinical testing.

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The Novel Synthetic Peptide (s<S) PSRCDCR-NH2, Reduces Carotid Artery Thrombus Formation In Rabbits By Inhibiting Platelet Aggregation

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Background: In acute coronary syndromes, platelet activation and aggregation are primarily mediated by GPIIb/IIIa receptors binding to their ligands, through the RGD (Arg-Gly-Asp) sequence.